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Canine and feline abdominal arterioportal communications can be classified based on branching patterns in computed tomographic angiography

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Abstract

Arterioportal vascular anomalies are communications between the splanchnic arteries and the portal system that represent a rare cause of presinusoidal portal hypertension in small animals. There is little information concerning the imaging findings of arterioportal communications in small animals and no classification could be found for radiologists and surgeons. The aims of this retrospective descriptive multicentric study were to describe the computed tomographic characteristics of arterioportal communications in a group of cats and dogs, and to propose a classification based on computed tomography (CT) angiographic anatomy. Computed tomography databases from multiple veterinary hospitals were searched for cats and dogs with a diagnosis of arterioportal communication. A total of 36 animals (33 dogs, three cats) met the inclusion criteria. There were 32 intrahepatic arterioportal malformations and four extrahepatic fistulae. The intrahepatic arterioportal malformations were classified as right divisional (11/32) and left divisional (21/32), and the left divisional were subclassified as left medial (16/21) and left lateral (4/21). One patient showed multiple intrahepatic arterioportal communications with concomitant left medial and left lateral conformations. Two patients with intrahepatic arteriovenous malformation showed concomitant congenital intrahepatic shunts. The proposed anatomical classification based on CT angiography could allow veterinary radiologists to have a more systematic approach and help improve the radiologist–surgeon communication.

KEYWORDS

arterioportal fistula, arterioportal malformation, congenital vascular anomaly

Swan Specchi and Federica Rossi contributed equally to this study.

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1 | INTRODUCTION

In people, vascular malformations are subdivided into two categories: slow-flow and fast-flow malformations. Slow-flow malformations represent anomalous connections among capillary, venous, and lymphatic components. High-flow vascular malformations contain arterial components in combination with other vascular structures.¹ Arteriovenous fistulae and arteriovenous malformations are both high-flow anomalies. Arteriovenous fistulae have a direct connection between the artery and vein without any intervening network and are usually acquired lesions secondary to trauma, surgical interventions, and neoplasia.^{2–6} Alternatively, arteriovenous malformations have a dense network of abnormal vessels representing the arteriovenous communication, called a “nidus”. Arteriportal vascular anomalies are communications between the splanchnic arteries and the portal system that represent a rare cause of presinusoidal portal hypertension in small animals.^{7,8}

Computed tomography (CT) angiography or selective fluoroscopic angiography allow direct visualization of the canine hepatic and related vasculature.^{7,8} Previously reported CT findings in dogs with arteriportal communication have included enlargement of the afferent artery, ascites (commonly), acquired porto-systemic shunts, aneurysmatic dilation of the portal vessels, and intrahepatic biliary ducts mineralizations.⁸ Fluoroscopy has been reported to allow better visualization of the actual flow through lesions and the communication between vessels. Potential treatment options for arteriovenous and arteriportal communications have included embolization or ligation of the dominant outflow vein, resection of one or more hepatic lobes, or transarterial embolization with cyanoacrylate glue.⁹ At the time of this study, no anatomic classification criteria for arteriportal communications could be found for use by veterinary radiologists and surgeons.

The aims of this study were to describe the CT imaging findings of arteriportal communications in a group of cats and dogs, and to propose a classification based on the CT angiographic anatomy.

2 | MATERIALS AND METHODS

This was a retrospective descriptive multicentric study. Medical electronic report databases of 12 veterinary hospitals (Animal Medical Center, the Clinica Veterinaria dell'Orologio, North Carolina State Veterinary Hospital, the Policlinico Veterinario Roma Sud, Royal Veterinary College Royal (Dick) School of Veterinary Studies of the University of Edinburgh, Hope Advanced Veterinary Hospital, Faculty of Veterinary Science of Chulalongkorn University, Clinica Veterinaria Santa Fara, Ospedale Veterinario Pingry, Alphavet, and École Nationale Vétérinaire d'Alfort) were reviewed to identify dogs with arteriportal communications diagnosed between 2007 and 2017. Dogs were included if abdominal CT angiography studies with both pre- and postcontrast images were available for review. Subject inclusion or exclusion decisions were made based on a consensus of two board-certified veterinary radiologists (S.S. and F.R.). All examinations were undertaken as part of clinical practice, and hospital directors approved use of data. Images were analyzed by two board-certified

veterinary radiologists (S.S. and F.R.) and an imaging intern (S.M.) through a two-step approach using a DICOM viewer software (Osirix DICOM viewer, Pixmeo, Geneva Switzerland). The collected data included contrast bolus direction when arterial and portal phases were available (performed through the evaluation of the attenuation in the cranial and caudal abdominal aorta and in the vessels of the cranial and caudal aspect of the portal system), localization of the arterioportal communication (as intra or extra-hepatic), identification of the afferent artery (or arteries) and efferent vein(s), change in diameter of the aorta caudal to the afferent artery, subjective enlargement of the afferent artery, aneurysmatic dilatation of the portal branches (defined as subjectively saccular or fusiform dilatation of the portal system), segmental or diffuse microhepatia, acquired portal collateral circulation, presence of indirect imaging findings of portal hypertension (ascites, pancreatic edema, gastric wall edema and gallbladder wall edema), biliary abnormalities (intra- or extrahepatic biliary ducts dilatation and/or cholelithiasis), and/or presence of other concomitant abdominal vascular abnormalities. References for normal abdominal vascular diameters are not reported in veterinary medicine. For this reason, the vascular diameter was subjectively classified as normal or increased based on previously reported qualitative criteria.⁸

In order to propose a computed tomographic classification for the anatomical appearance of arteriportal communications, images were reviewed multiple times looking for repetitive anatomical patterns until observers reached a consensus on anatomical-based classification criteria. When available, the presence of portal hypertension through direct invasive catheterization of the portal vein was recorded. For inclusion in this study, portal hypertension had to have been measured using a 4Fr Cobra catheter, a 0.035” angled hydrophilic guide wire combination (Infiniti Medical, LLC, Redwood City, CA) and fluoroscopic guidance to selectively access the shunt and/or portal vein. Portal pressure was recorded through the end-hole of a Cobra catheter.

3 | RESULTS

Thirty-six patients were included (12 from Animal Medical Center, six from the Clinica Veterinaria dell'Orologio, four from North Carolina State University Veterinary Hospital, three from the Policlinico Veterinario Roma Sud, three from the Royal (Dick) School of Veterinary Studies, two from Royal Veterinary College, one from Hope Advanced Veterinary Hospital, one from the Faculty of Veterinary Science of Chulalongkorn University, one from Clinica Veterinaria Santa Fara, one from Ospedale Veterinario Pingry, one from Alphavet, and one from the École Nationale Vétérinaire d'Alfort).

There were 33 dogs (four intact females, 11 spayed females, 10 intact males, and eight neutered males) and three cats (two spayed females and one male neutered). Mean age was 30 months (range from 3 to 108 months). Breeds included were 17 mixed breed dogs, five Labrador Retrievers, three Welsh Corgie, two Poodle, two Weimaraner, one West Highland White Terrier, one Beagle, one Golden Retriever, and one Great Dane. The three cats were all domestic shorthair.

Descriptions of imaging equipment and technical parameters used for CT scanning at each of the hospitals are provided in Appendix. A CT arterial phase was available in 21/36 patients. There were 32 intrahepatic and four extrahepatic arteriportal communications. In the intrahepatic arteriportal communications, a clear identification of the afferent vessel was possible in only 13/32 cases while the efferent vessels were identified in all patients. Hepatofugal direction of the contrast bolus in the portal system was observed in 26/36 patients demonstrated by the presence of contrast medium in the cranial mesenteric vein during the arterial phase without contrast in the jejunal vein or caudal mesenteric vein. In addition, in these patients, there was equal distribution of the contrast medium bolus in the aorta and cranial mesenteric vein and/or its tributaries during the arterial phase.

Other imaging findings included subjective enlargement of the afferent artery (23/36), aneurysmatic dilatation of the portal branches (21/36), decreased diameter of the abdominal aorta caudal to the afferent artery (19/36), and segmental (9/36) or diffuse (15/36) microhepatia.

Multiple patterns of acquired portal collateral circulation were observed in 34/36 patients and gallbladder varices were observed in 12/36 patients. The two patients without collateral portal circulation had concomitant congenital single intrahepatic shunts.

Indirect imaging features of portal hypertension were pancreatic edema in 20/36, ascites in 20/36, and gastric wall edema 19/36. Invasive evaluation of portal pressure was available in 12 patients with hepatofugal bolus dynamic in the portal system and confirmed portal hypertension in all of them.

Biliary abnormalities were observed in six of 36 patients. Two patients had intrahepatic cholelithiasis, one patient had subjective dilatation of the gallbladder, one patient had cholelithiasis in the gallbladder and subjective dilatation of the gallbladder, one patient had dilatation of the intrahepatic biliary ducts in the quadrate lobe, and one patient had subjective distention of the common bile duct.

Due to the inconsistent visualization of the afferent vessel, we classified the intrahepatic arteriportal communications only based on the efferent vessel in left (21/32) and right (11/32) (Figure 1) divisions. The left-divisional communications were subclassified according to the corresponding portal branch into left medial (if the quadrate or right medial were involved [16/21]) (Figure 2) and left lateral (if the left lateral or left medial were involved [4/21]) (Figure 3). One patient showed multiple intrahepatic arteriportal communications with a concomitant left medial and left lateral conformation (Figure 4). Right-divisional communications always involved the right lateral portal vein (11/11) and in two cases, concomitant dilatation of the caudate portal branch was observed. For this reason, a sub-classification for the right-divisional was not possible. In case of intrahepatic left-divisional arteriovenous malformations and medial conformation, enlargement of the portal branches of both right medial and quadrate lobe were observed in six of 16 patients. These branches were in continuity with the gallbladder varices (Figure 5). Only animals with left-divisional and medial intrahepatic arteriportal communications had gallbladder varices.

In the four of 20 cases of intrahepatic left-divisional and lateral conformation, only one case showed clear involvement of the portal

vessel of the left medial hepatic lobe with normal conformation of the portal vessel of the left lateral hepatic lobe. In the other three cases of intrahepatic left-divisional and lateral conformation, the arteriportal malformation involved the portal vessels of both left lateral and medial hepatic lobes.

See Table 1 for a schematic classification of single versus double efferent veins in left and right-divisional arteriportal communication. In 20 cases, a single intrahepatic portal branch was involved, including 11 left-divisional and nine right-divisional communication. In three patients with extrahepatic arteriportal communications, the afferent artery was represented by the caudal mesenteric artery or one of its branches. The efferent veins were the cranial mesenteric vein or the left colic vein. One dog had two extrahepatic arteriportal communications, a more cranial arteriportal communication with the afferent artery consistent with the distal portion of the cranial mesenteric artery and the efferent vein was identified as the cranial mesenteric vein. A segmental saccular aneurysmatic dilatation of the caudal portion of the cranial mesenteric vein was also observed. The afferent artery of the caudal arteriovenous communication was the caudal mesenteric artery and the efferent vessel was the left colic vein. One dog with extrahepatic arteriportal communication showed also multiple healed pelvic fractures.

Other concomitant vascular abnormalities that were identified were a congenital left-divisional intrahepatic porto-systemic shunt in two of 36 patients with a right-divisional and left-divisional medial arteriportal communication. In patients with concomitant intrahepatic shunt contrast medium was visible in the intra- and posthepatic tract of the caudal vena cava during the arterial phase. Indirect imaging features of portal hypertension were absent in these patients.

4 | DISCUSSION

Computed tomographic features of canine and feline arteriportal communications are reported in this study and a classification based on the CT angiographic anatomy is proposed. In this cohort of dogs and cats, intrahepatic arteriportal communications were more common than the extrahepatic arteriportal communication. Computed tomographic visualization of the afferent and efferent vessels was variable. In patients with intrahepatic arteriportal communications, the branch of the hepatic artery responsible for the arteriportal communication was inconstantly visualized. In patients with extrahepatic arteriportal communication, there was no visible "nidus" and the artery responsible for the arteriportal communication was always identified. In the authors' opinion, there are two different factors influencing the ability to determine the afferent artery: the complexity of the anatomical conformation of the hepatic artery compared to the cranial and caudal mesenteric arteries and the type of arteriportal communication. In particular, intrahepatic arteriportal communications always showed a nidus, with presence of a multitude of adjacent and tortuous vessels making the identification of the afferent artery difficult. In contrast, the extrahepatic arterio-portal communication did not show any nidus, allowing an easier identification of the direct communication between the afferent artery and efferent vein. For these reasons, the term

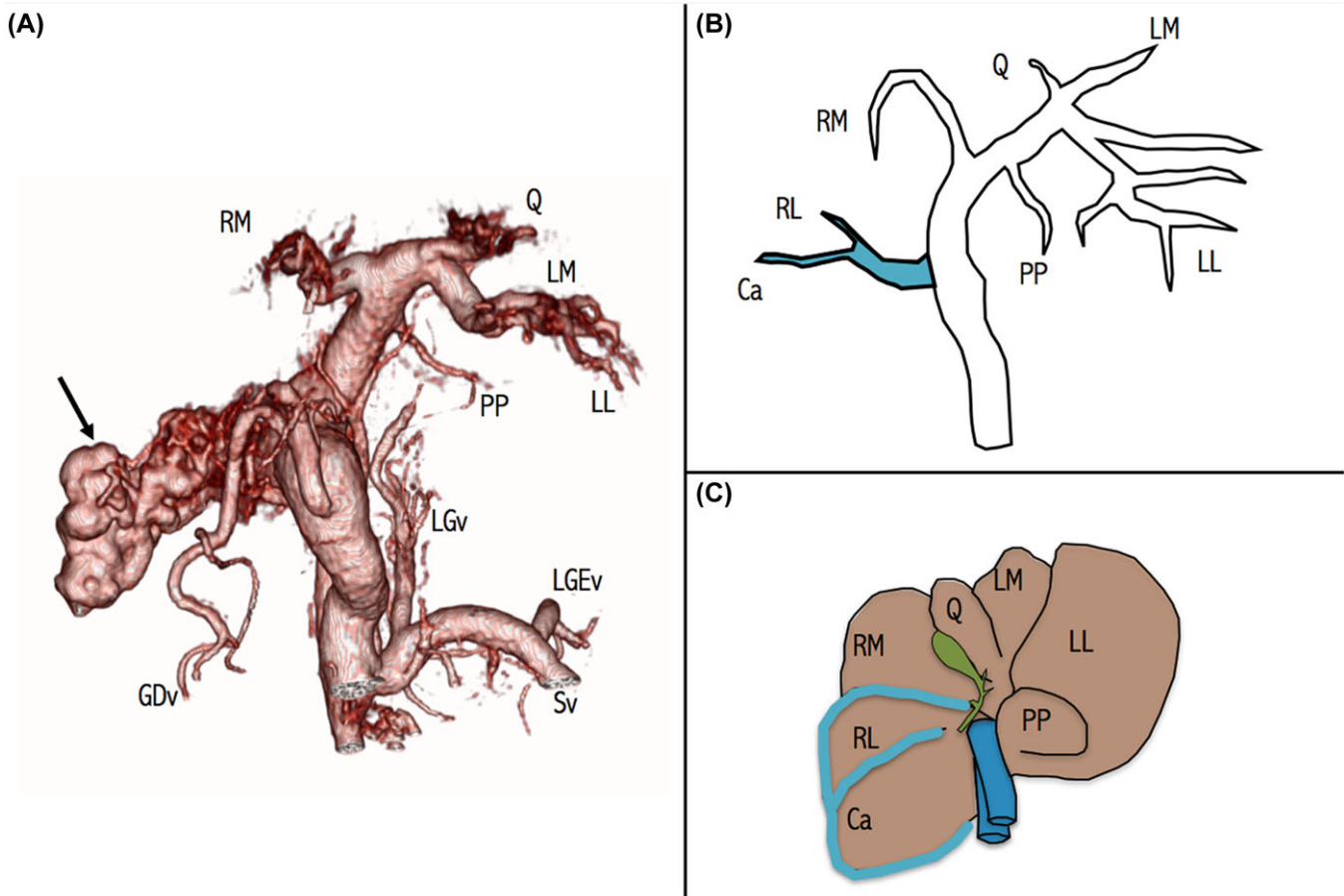


FIGURE 1 Three-dimensional volume-rendered computed tomography image illustrating the arterial phase of a dog with intrahepatic right divisional arteriportal malformation (A). Note the dense network of tortuous vessels (black arrow) representing the “nidus”. Schematic representation of the portal branches (B) and hepatic lobes (C) involved in the intrahepatic right divisional arteriportal malformation. A and B, RM, right medial portal branch; Q, quadrate lobe portal branch; LM, left medial lobe portal branch; LL, left lateral lobe portal branch; PP, papillary process portal branch; RL, right lateral portal branch; Ca, caudate lobe portal branch. C, RM, right medial hepatic lobe; Q, quadrate hepatic lobe; LM, left medial hepatic lobe; LL, left lateral hepatic lobe; PP, papillary process; RL, right lateral hepatic lobe; Ca, Caudate hepatic lobe. A, LGv, left gastric vein; LGEv, left gastro-epiploic vein; Sv, splenic vein; GDv, gastroduodenal vein [Color figure can be viewed at wileyonlinelibrary.com]

arteriportal malformations is more appropriate when referring to intrahepatic arteriportal communications while arteriportal fistula is a more suitable term for extrahepatic arteriportal communication. Even if there was no evidence of a previous trauma or surgery, it was not possible to finally confirm the congenital nature of the intrahepatic communication in our cases. However, the young age of the patients, the repetitive anatomical patterns, and the presence of a nidus make the hypothesis of congenital intrahepatic arteriportal malformations most likely. Interestingly, one of the four dogs with extrahepatic fistula, presented healed pelvic fractures suggesting a possible acquired origin.

All patients with intrahepatic right-divisional arteriportal malformation showed involvement of the portal vein of the right lateral hepatic lobe. Concomitant involvement of the portal vein of the caudate lobe was observed in two patients. This is a useful information for surgeons approaching this group of patients because vascular occlusion of the main right portal branch may allow complete closure of the arteriportal malformation.

In patients with arterial phase available, we detected the same bolus dynamic in the aortic and portal circulation (portal vein and

cranial mesenteric vein). This finding demonstrates that in the arterial phase the bolus direction in the aorta and portal vein/cranial mesenteric vein has similar dynamics suggesting a hepatofugal direction of the blood flow in the cranial mesenteric vein. Furthermore, the presence of severe portal hypertension was confirmed through invasive catheterization of the portal vein in 12 patients. Based on the authors personal experience, the intrahepatic arteriportal malformations have hepatofugal portal blood flow unless a concurrent intrahepatic portosystemic shunt is present.

As previously reported, we commonly observed an increased size of the afferent artery.⁸ This is due to low resistance to blood flow through the arteriportal communication compared to the blood flow resistance in arteries, capillaries, and hepatic sinusoids with secondary increased blood volume through the afferent vessel over time. There is also “siphoning” of blood from the nearby normal vessels into the arteriportal communication contributing to the enlargement of the afferent artery and with secondary decreased oxygenation of the adjacent organs, also called “blood steal phenomenon”.^{8,10–14} The “blood steal phenomenon” causes decreased blood flow in the arteries adjacent to the arteriportal communication explaining the abrupt decreased

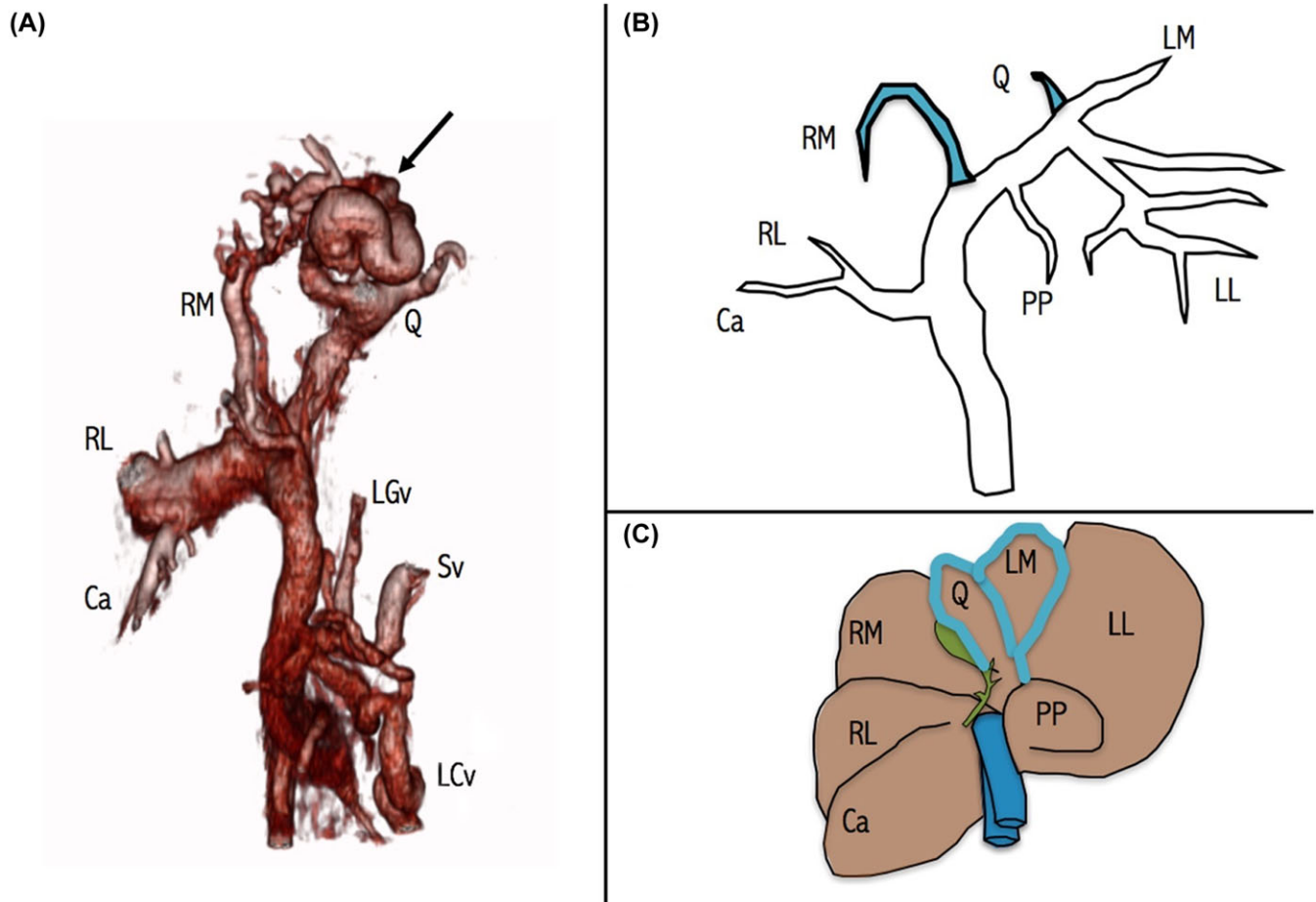


FIGURE 2 Three-dimensional volume-rendered CT image illustrating the arterial phase of a dog with intra-hepatic left divisional and medial arterioportal malformation (A). Note the dense network of tortuous vessels (black arrow) representing the “nidus”. Schematic representation of the portal branches (B) and hepatic lobes (C) involved in the intra-hepatic left divisional and medial arterioportal malformation. A and B, RM, right medial portal branch; Q, quadrate lobe portal branch; LM, left medial lobe portal branch; LL, left lateral lobe portal branch; PP, papillary process portal branch; RL, right lateral portal branch; Ca, caudate lobe portal branch. C, RM, right medial hepatic lobe; Q, quadrate hepatic lobe; LM, left medial hepatic lobe; LL, left lateral hepatic lobe; PP, papillary process; RL, right lateral hepatic lobe; Ca, caudate hepatic lobe. A, LGv, left gastric vein; Sv, splenic vein; LCv, left colic vein [Color figure can be viewed at wileyonlinelibrary.com]

diameter of the aorta and cranial mesenteric artery observed in this study and as previously reported by Zwingenberger et al.⁸

Aneurysmatic dilatation of the portal vein and its branches was a common finding in patients with intrahepatic arterioportal malformations. Aneurysmatic dilatation of the cranial mesenteric vein was also observed in a patient with extrahepatic arterioportal fistula. Aneurysmatic dilatation of the portal vein and of its intrahepatic branches has been previously reported alone¹⁵ or associated to other concomitant vascular diseases.^{8,16–18} As reported in humans, aneurysmatic dilatation of the portal vein and its branches has likely resulted from chronic portal hypertension and turbulent arterial flow with secondary weakening of the wall with progressive thickening of the intima and replacement by fibrous tissues.^{19–21}

Ascites was a frequent finding. Factors that contribute to ascites formation are portal hypertension and hypoproteinemia. Portal pressure was invasively evaluated in 12/35 patients confirming the presence of portal hypertension. We did not retrospectively evaluate the total proteins and albumin levels in these patients. However, we presume that the association of arterial “blood steal

phenomenon” and portal hypertension may have caused venous congestion of the gastrointestinal tract with secondary malabsorption in the small bowel and possible hypoproteinemia as previously reported.⁸

Presence of microhepatia was a frequent finding. We presumed microhepatia was related to the decreased amount of blood reaching the hepatocytes due to “blood steal phenomenon” in the hepatic artery and portal hypertension with hepatofugal flow. With “blood steal phenomenon” the arterial blood bypasses the arterial capillaries with decreased amount of oxygen to the hepatocytes.⁸ The presence of portal hypertension, similar contrast bolus dynamic in the aorta and portal vein during the arterial phase and acquired porto-systemic shunt suggest hepatofugal flow that may also have contributed to decreased oxygen and nutrients to hepatocytes. Multiple extrahepatic arterioportal fistulae were observed in one patient. The presence of multiple concomitant vascular anomalies is related to the common embryological origin of these vessels, and developmental abnormalities may affect multiple vessels as previously reported.²² One patient with extrahepatic arterioportal fistula had multiple healed

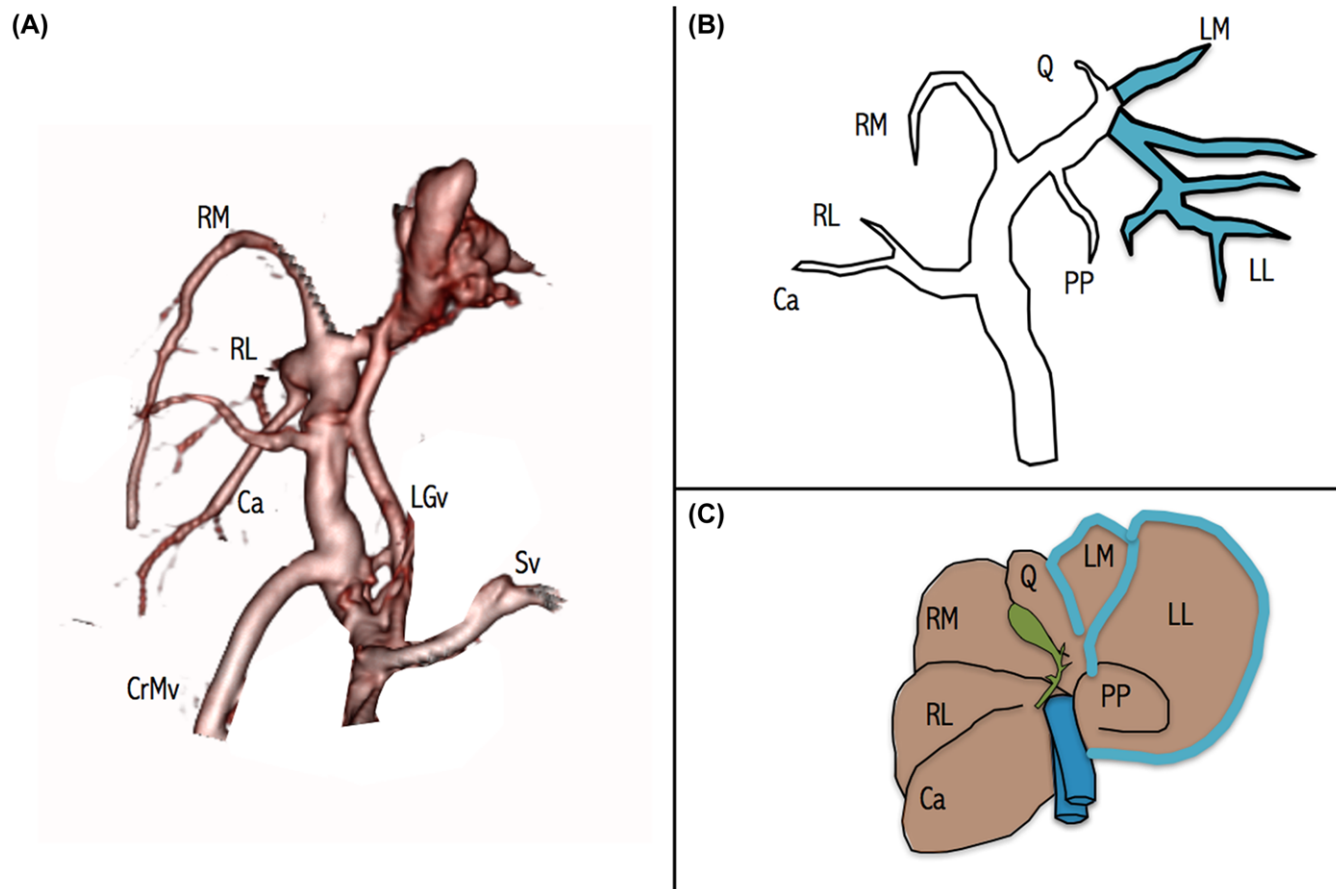


FIGURE 3 Three-dimensional volume-rendered computed tomography image illustrating the arterial phase of a dog with intra-hepatic left divisional and lateral arterioportal malformation (A). Note the dense network of tortuous vessels (black arrow) representing the "nidus". Schematic representation of the portal branches (B) and hepatic lobes (C) involved in the intrahepatic left divisional and lateral arterioportal malformation. A and B, RM, right medial portal branch; Q, quadrate lobe portal branch; LM, left medial lobe portal branch; LL, left lateral lobe portal branch; PP, papillary process portal branch; RL, right lateral portal branch; Ca, caudate lobe portal branch. C, RM, right medial hepatic lobe; Q, quadrate hepatic lobe; LM, left medial hepatic lobe; LL, left lateral hepatic lobe; PP, papillary process; RL, right lateral hepatic lobe; Ca, Caudate hepatic lobe. A, LGv, left gastric vein; Sv, splenic vein; CrMv, cranial mesenteric vein [Color figure can be viewed at wileyonlinelibrary.com]

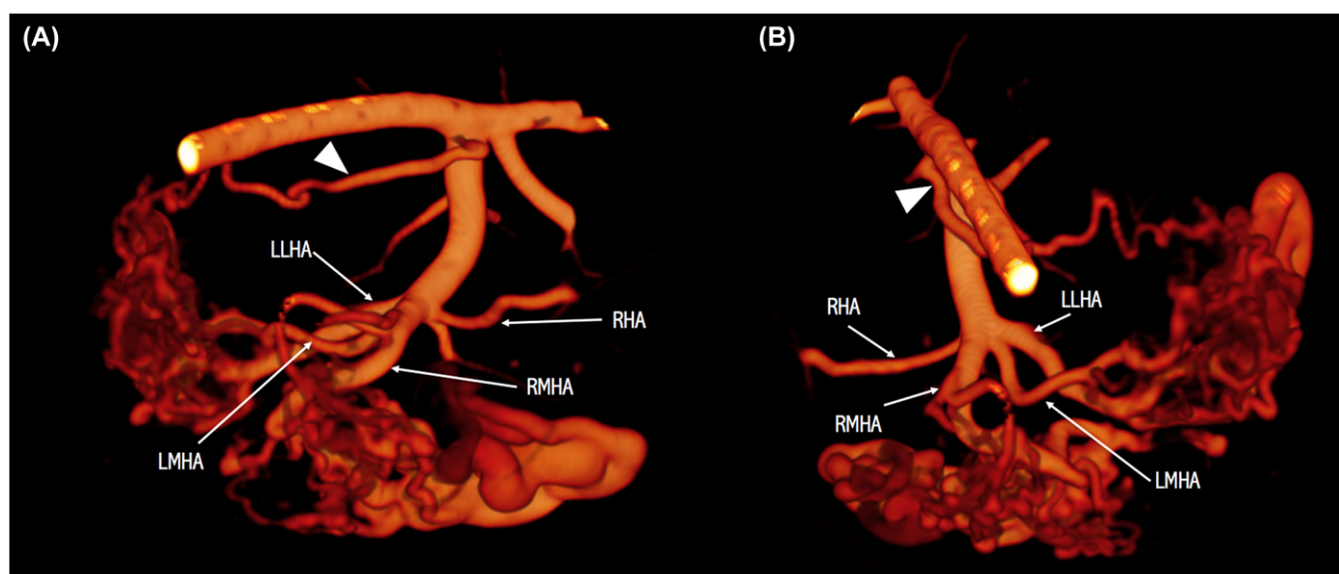


FIGURE 4 Three-dimensional volume-rendered computed tomography image illustrating the arterial phase of a dog with intrahepatic multiple arterioportal communication with concomitant left divisional and medial and left divisional and lateral conformation. In image (A) cranial is to the left. In image (B) right is to the left. Note the enlargement diaphragmatic artery (arrow head). RHA, right hepatic artery; LLHA, left lateral hepatic artery; LMHA, left medial hepatic artery; RMHA, right medial hepatic artery [Color figure can be viewed at wileyonlinelibrary.com]

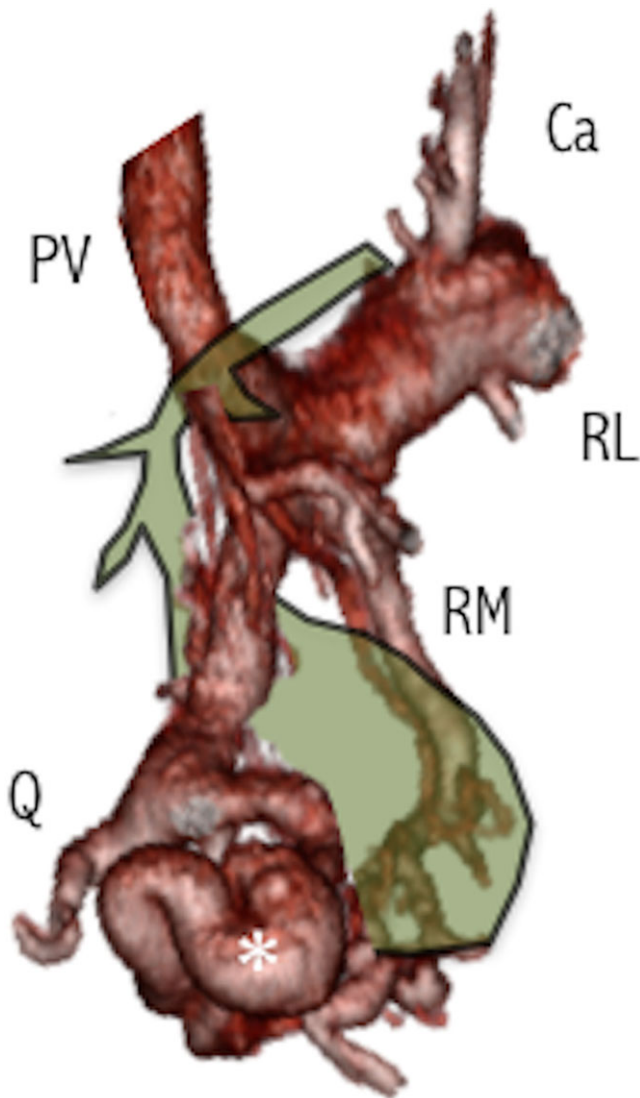


FIGURE 5 Three-dimensional volume-rendered computed tomography image illustrating the arterial phase of a dog with intrahepatic left divisional and medial arterioportal malformation superposed to schematic draw of the gallbladder. Note the enlargement of the portal branches of the quadrate and right medial lobe communicating between them at the level of the arterioportal malformation (asterisk) [Color figure can be viewed at wileyonlinelibrary.com]

pelvic fractures. It is possible that in this patient the fistula is acquired post-traumatic as reported in humans.³

Variable patterns of portal collateral circulation have been observed in our cohort of dogs reflecting the previously proposed

classification.¹⁶ We observed gallbladder varices only in patients with arterioportal malformations with an intrahepatic left-divisional and medial conformation. Interestingly, also in a previous study concerning acquired portal collateral circulation due to different causes, gallbladder varices were observed only in a patient with an intrahepatic arterioportal malformation.¹⁶ The arterial perfusion of the gallbladder has been previously described as originating from the left medial branch of the left hepatic artery or directly from the left hepatic artery.^{23,24} The right medial branch of the hepatic artery is also responsible for the perfusion of the right medial lobe, dorsal portion of the quadrate lobe, and part of the left medial lobe.²⁵ Little information is available concerning the venous drainage of the gallbladder wall in small animals. In humans, the gallbladder wall drainage consists in multiple cystic/cholecystic veins that drain mainly within the portal system to subsequently join the middle or right hepatic veins.²⁶ We observed enlargement of the portal branches of the right medial and quadrate lobes in patients with gallbladder varices and intrahepatic left-divisional and medial arterioportal malformation. Our hypothesis is that gallbladder varices in these patients do not represent collateral portal circulation but enlargement of the venous and arterial capillary bed due to direct involvement of these vessels in the arterioportal malformation nidus with secondary increased blood flow. The enlargement of the portal branches of the right medial and quadrate lobe suggests that these branches are responsible for the gallbladder wall venous drainage in these patients.

Gallbladder, intra- and extrahepatic biliary tract distension, and cholelithiasis of the gallbladder were observed. Biliary ischemia due to arterioportal shunt is recognized in human as cause of biliary dilatation, biliary cyst, or strictures.²⁷ Our hypothesis is that these conditions may have predisposed to biliary tract distention and cholelithiasis of the gallbladder. In dogs with portosystemic shunts, arteriolar proliferation and biliary hyperplasia have been reported and could also play a role in the development of biliary abnormalities.²⁸

Arterioportal malformation with concomitant congenital intrahepatic portosystemic shunting was detected in two patients with no ascites and no portal collateral circulation. We believe that the absence of ascites in these patients is due to the arterial blood entering the caudal vena cava directly, bypassing the portal system, with lack of portal hypertension signs compared to patients with arterioportal malformation only.

The main limitations of the study were the different computed tomographic angiography protocols, the absence of arterial phase, and the invasive evaluation of portal pressure in only a portion of patients. Furthermore, conventional CT does not offer the advantages

TABLE 1 Classification of 32 intrahepatic arterioportal communications

| | Left Lateral | | Left Medial | | Right | | Total |
|-----------------------|--------------|----|-------------|----|-------|----|-------|
| | LL | LM | Q | RM | Cd | RL | |
| Single efferent vein | 0 | 1 | 4 | 6 | 0 | 9 | 20 |
| Double efferent veins | | 3 | | 6 | | 2 | 11 |
| Multiple | | | 1 | | 0 | | 1 |
| Total | | 4 | | 16 | | 11 | 32 |

Note. LL, left lateral; LM, left medial; Q, quadrate; RM, right medial; Cd, caudate; RL, right lateral.

of complete dynamic as provided by digital fluoroscopy, therefore the representation of the abnormal vascular anatomy might be incomplete.

The consensus on the classification of the arteriportal communication was the following: patients with intrahepatic arteriportal malformations could be classified based on the efferent portal vein (or veins) as left or right lateral divisional and in case of left lateral divisional, they could be subclassified as medial versus lateral. Patients with extrahepatic arteriportal communications could be classified based on the name of the afferent and efferent vessel only.

There are still no clear guidelines in the treatment of arteriportal malformations, but different possibilities have been proposed such as embolization of the afferent vessel or liver lobectomy.⁷ Future promising treatments such as outflow vein embolization are recently proposed also in veterinary medicine and these techniques would benefit from an outflow portal vein classification scheme.²⁹ In order to facilitate the radiologist–surgeon communication, to help the presurgical planning and to support the treatment choice, we decided to emphasize the name of the afferent and efferent vessel (in case of extrahepatic arteriportal malformation) or the lobe/lobes in which the efferent portal vessel (and number of efferent vessels) was located (in case of intrahepatic arteriportal malformation).

In conclusion, findings from the current study describe detailed computed tomographic features of arteriportal malformations in a sample of small animals from multiple veterinary hospitals. The authors also proposed an anatomical classification system based on CT angiography characteristics that could allow veterinary radiologists to have a more systematic approach and help improve radiologist–surgeon communications.

LIST OF AUTHOR CONTRIBUTIONS

Category 1

- (a) Conception and Design: Specchi S, Rossi F, Weisse C
- (b) Acquisition of Data: Specchi S, Rossi F, Weisse C, Morabito S, Petrovitch NP, Drees R, Thierry F, Ricciardi M, Penchome R, Armenise A, Pey P, Paek M, Panopoulos I, Nicoli S, Schwarz T
- (c) Analysis and Interpretation of Data: Specchi S, Rossi F, Weisse C, Morabito S

Category 2

- (a) Drafting the Article: Specchi S, Rossi F, Morabito S, Weisse C
- (b) Revising Article for Intellectual Content: Specchi S, Rossi F, Weisse C, Morabito S, Petrovitch NP, Drees R, Thierry F, Ricciardi M, Penchome R, Armenise A, Pey P, Paek M, Panopoulos I, Nicoli S, Schwarz T

Category 3

- (a) Final Approval of the Completed Article: Specchi S, Rossi F, Weisse C, Morabito S, Petrovitch NP, Drees R, Thierry F,

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APPENDIX: DESCRIPTION OF IMAGING EQUIPMENT AND TECHNICAL PARAMETERS USED FOR CT SCANNING AT EACH OF THE HOSPITALS

| Institution | Imaging equipment | Technical parameters |
|-----------------------------------|---|--|
| Clinica Veterinaria dell'Orologio | 16-Row MDCT unit (Light Speed 16 slices, GE Medical Systems, Milan, Italy) | Images were acquired in helical scan mode, at 120 kV and 160–210 mAs tube settings, a pitch of 0.562:1 and 1.25 mm slice thickness with 50% overlap with a 0.7 s rotation time and reconstructed with a non-enhancing non-smoothing algorithm. Contrast-enhanced images were obtained using a dosage of 2.2 mL/kg of Ioversol (Optiray 300 mg I/mL, Covidien, Segrate, MI, Italy). |
| Clinica Veterinaria Santa Fara | 16-Row MDCT unit (Light Speed 16 slices, GE Medical Systems, Milan, Italy) | Images were acquired in helical scan mode, with a pitch of 0.56 slice thickness of 1.25 mm, 0.7 s tube rotation time, 120 kV, and 180 mAs. Contrast-enhanced images were obtained using a dosage of 1.5 mL/kg of Ioversol (Optiray 350 mg I/mL, Mallinckrodt Pharmaceuticals, Segrate, Italy). |
| Ospedale Veterinario Pingry | 16-Row MDCT unit (Somatom Emotion, Siemens, Forchheim, Germany) | Images were acquired in helical scan mode, at 180 kV and 110 mAs tube settings, using a pitch of 0.8, 1 mm slice thickness and 0.6 s tube rotation time. Contrast-enhanced images were obtained using a dosage of 2 mL/kg of Iopamidol (Iopamigita 370 mg I/mL, Agfa H.C. Imaging Agents GmbH, Sweden). |
| Royal Veterinary Collage | 16-Row MDCT unit (Philips MX8000 IDT, Phillips Healthcare Andover, Massachusetts) | Cat: images were acquired in helical scan mode, with a pitch of 1.25, slice thickness of 2 mm, 0.7 s tube rotation time, 90 kV, and 150 mAs. Dog: images were acquired in helical scan mode, with a pitch of 1.25, slice thickness of 2 mm, 0.7 s tube rotation time, 120 kV, and 199 mAs. Contrast-enhanced images were obtained using a dosage of 2 mL/kg of Iohexol (Omnipaque 350 mg I/mL, Nycomed, Oslo, Norway). |

(Continued)

| Institution | Imaging equipment | Technical parameters |
|--|---|--|
| Small Animal Teaching Hospital, Faculty of Veterinary Science, Chulalongkorn | 64-Row MDCT unit (Light Speed 64 slices, GE Medical Systems, Milan, Italy) | <p>Cat: images were acquired in helical scan mode, with a pitch of 0.65, slice thickness of 1.25 mm, 0.6 s tube rotation time, 100 kV, and 600 mAs</p> <p>Dog: images were acquired in helical scan mode, with a pitch of 1.25, slice thickness of 1.25 mm, 0.6 s tube rotation time, 100 kV, and 600 mAs.</p> <p>Contrast-enhanced images were obtained using a dosage of 2 mL/kg of Iopamidol (Iopamigita 370 mg I/mL, Agfa H.c. Imaging Agents GmbH, Sweden).</p> |
| Policlinico Veterinario Roma SUD | 16-Row MDCT unit (Philips MX16, Phillips Healthcare Andover, Massachusetts) | <p>Cat: images were acquired in helical scan mode, with a pitch of 1, slice thickness of 1 mm, 0.6 s tube rotation time, 120 kV, and 190 mAs</p> <p>Dog: images were acquired in helical scan mode, with a pitch of 2, slice thickness of 2 mm, 0.6 s tube rotation time, 120 kV, and 190 mAs.</p> <p>Contrast-enhanced images were obtained using a dosage of a dosage of 2 mL/kg of Iohexol (Omnipaque 350 mg I/mL, GE healthcare S.r.l., Milan, Italy).</p> |
| University of Edinburgh teaching hospital | 4-Row MDCT unit (Somatom Volume Zoom, Siemens, Germany) | <p>The plain transverse images of the abdomen were acquired in helical mode using the following scanning parameters: 3 mm slice thickness, tube current of 100–180 mA, tube voltage of 120 kVp and 512 × 512 matrix.</p> <p>Contrast-enhanced images were obtained using a dosage of 2 mL/kg of Iopamidol (Niopam 370 mg I/mL, Bracco, Patheon Italia S.p.A., Ferentino (FR), Italy).</p> |
| Alphavet | 16-Row MDCT unit (Light Speed 16 slices, GE Medical Systems, Milan, Italy) | <p>The images were acquired in helical scan mode, at 140 kV and 150–200 mAs tube settings, a pitch of 0.562:1 and 1.25 mm slice thickness with a 0.7 s rotation time.</p> <p>Contrast-enhanced images were obtained using a dosage of 2 mL/kg of Iobitridol (Xenetix 350 mg I/mL, Guerbet, France).</p> |
| NC State Veterinary Hospital | 64-Row MDCT unit (Somatom Emotion, Siemens, Forchheim, Germany) | <p>The images were acquired in helical scan mode, at 120–130 kV, 500–200 mAs and 1 mm slice thickness.</p> <p>Contrast-enhanced images were obtained using a dosage of 4 mL/kg of Iohexol (Omnipaque 350 mg I/mL, Nycomed Inc., Princeton, NJ 08540).</p> |
| Alfort Veterinary School (France) | 16-Row MDCT unit (General Electric, GE Bright, Speed 16 TM) | <p>The images were acquired in helical scan mode, at 100 kV, 250 mAs, a pitch 0.625, 1.5 mm slice thickness and rotation time 0.5 sec.</p> <p>Contrast-enhanced images were obtained using a dosage of 2 mL/kg of Iopamidol (Iopamiron 300 mg I/mL, Bracco Imaging France, Courcouronnes, France).</p> |